

Elevated Blood Lead Levels in Children of Construction Workers

ABSTRACT

Objectives. This study examined whether children of lead-exposed construction workers had higher blood lead levels than neighborhood control children.

Methods. Twenty-nine construction workers were identified from the New Jersey Adult Blood Lead Epidemiology and Surveillance (ABLES) registry. Eighteen control families were referred by workers. Venous blood samples were collected from 50 children (31 exposed, 19 control subjects) under age 6.

Results. Twenty-six percent of workers' children had blood lead levels at or over the Centers for Disease Control and Prevention action level of 0.48 $\mu\text{mol/L}$ (10 $\mu\text{g/dL}$), compared with 5% of control children (unadjusted odds ratio = 6.1; 95% confidence interval = 0.9, 147.2).

Conclusions. Children of construction workers may be at risk for excessive lead exposure. Health care providers should assess parental occupation as a possible pathway for lead exposure of young children. (*Am J Public Health*. 1997;87:1352-1355)

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Introduction

An increased understanding of the adverse effects of pediatric lead poisoning led the Centers for Disease Control and Prevention (CDC) to lower the level at which blood lead poses a health concern three times in the last 20 years.¹ The CDC now defines the pediatric blood lead level of concern as 0.48 $\mu\text{mol/L}$ (10 $\mu\text{g/dL}$) or higher. The percentage of US children aged 1 through 5 years with blood lead levels 0.48 $\mu\text{mol/L}$ (10 $\mu\text{g/dL}$) or greater dropped from 88.2% in 1976 through 1980 to 8.9% in 1988 through 1991, a remarkable public health achievement.² However, high rates of lead poisoning are still found in specific populations (i.e., Black, low-income, and urban children), and approximately 1.7 million children have blood lead levels of 0.48 $\mu\text{mol/L}$ (10 $\mu\text{g/dL}$) or higher.³

The objective of the current study was to examine an additional population at high risk for pediatric lead poisoning—the young children of lead-exposed construction workers. Construction workers may be exposed to dangerously high levels of lead dust,⁴ and employers were only recently required by the Occupational Safety and Health Administration (OSHA) to follow the same lead-exposure regulations as general industry.⁵ Because of the short-term and transient nature of many construction job sites, proper hygiene programs may not be available to minimize the potential for workers to inadvertently take lead home on their skin and clothing and in their vehicles.

Methods

Study Population and Data Collection

In 1994, workers in construction-related Standard Industrial Classification codes⁶ were identified from the New Jersey Adult Blood Lead Epidemiology and Surveillance (ABLES) registry, a statewide surveillance program that includes reports of New Jersey workers with blood lead levels of 25 $\mu\text{g/dL}$ or greater.⁷ Eligible workers were those who

(1) had been employed in construction for at least 1 month during the preceding year, (2) had at least one child under age 6 living in the household, and (3) were living at home while working in construction. Families of ineligible workers were offered free lead screening by the New Jersey State Department of Health.

To account for the effects of background lead exposures from such sources as paint in older homes, drinking water, and industrial emissions, neighborhood control households were chosen for each exposed household. Workers were asked to provide the name of one or two families in their neighborhood with at least one child between 9 months and 6 years of age. Control households were considered ineligible if any family member worked in a lead-related industry.

Personal interviews were conducted with the adult who had primary responsibility for child care in the household to obtain information about the household. Interviews were also conducted with each worker to obtain specific information about job characteristics and work practices. Venous blood samples were collected from all children who had not yet reached their sixth birthday. Samples of dust, loose paint chips, and water at exposed and control homes were collected for determination of lead concentrations. The methods for collection and analysis of these samples are described separately.⁸

Blood lead testing was performed at the New Jersey State Department of Health Laboratory in Trenton, NJ. Blood

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lead determinations were made by graphite furnace atomic-absorption spectrophotometry, and erythrocyte protoporphyrin levels were determined by hemofluorometric techniques. The lower limit of detection for blood lead determination was 0.05 $\mu\text{mol/L}$ (1.0 $\mu\text{g/dL}$).

Data Analysis

To assess the effect of exposure status on continuous measures of child blood lead, we derived mean blood lead levels and used *t* tests to compare the mean blood lead levels in the exposed and unexposed groups. Values for blood lead levels were log-transformed to correct skewness in the distribution. Logistic regression was used to model the association between exposure and an elevated blood lead level, defined as 0.48 $\mu\text{mol/L}$ (10 $\mu\text{g/dL}$) or greater, while potential confounders were controlled for. Because the number of study participants was small, exact inference methods were used to calculate odds ratios and 95% confidence intervals.^{9,10} The covariates considered as potential confounders were age and sex of the child, race and education level of the head of household, age of the home, percentage of lead in household paint, level of lead in drinking water, lead-related hobbies of any household member, home remodeling in the past year, use of imported ceramics to store/serve food, consumption of food from a household garden (since soil lead may be high), consumption of canned foods, and presence of a pet that spends time indoors and outdoors.

Because of the lack of independence associated with blood lead measurements from multiple children in the same household, we performed additional analyses using a class of generalized estimating equations to take into account the correlation of outcomes for members of the same household and to adjust effect estimates and their corresponding standard errors.¹¹

Correlations between measures of lead in dust and in child's blood were estimated by means of Pearson correlation coefficients. Log-transformed values were used for correlation measures.

Results

A total of 516 construction workers were initially identified as potential study participants from the New Jersey ABLES registry. We were unable to contact 210 of these workers (40%) to administer the telephone screening questionnaire, primar-

TABLE 1—Blood Lead and Erythrocyte Protoporphyrin Levels of Children under Age 6, by Exposure Status, New Jersey Adult Blood Lead Epidemiology and Surveillance (ABLES) Registry

	Exposed Children (n = 31)	Control Children (n = 19)
Blood lead, $\mu\text{mol/L}$ ($\mu\text{g/dL}$)		
Arithmetic mean ^a	0.36 (7.4)	0.25 (5.1)
Geometric mean ^a	0.29 (6.1)	0.22 (4.6)
Range	0.05–0.86 (1.0–17.9)	0.09–0.52 (1.8–10.7)
SD	0.22 (4.5)	0.12 (2.5)
Geometric SD	0.10 (2.0)	0.08 (1.6)
Percentage $\geq 10 \mu\text{g/dL}$	25.8	5.3
Odds ratio (95% CI)	6.1 (0.9, 147.2)	1.0
Erythrocyte protoporphyrin, $\mu\text{mol/L}$ ($\mu\text{g/dL}$)		
Arithmetic mean ^b	0.27 (15.1)	0.29 (16.3)
Geometric mean ^b	0.25 (14.3)	0.28 (15.7)
Range	0.16–0.50 (9.0–28.0)	0.16–0.46 (9.0–26.0)
SD	0.09 (5.3)	0.08 (4.5)
Geometric SD	0.02 (1.4)	0.02 (1.3)

Note. CI = confidence interval.

^a*P* = .11 (*t* test on log-transformed values).

^b*P* = .30 (*t* test on log-transformed values).

ily because correct telephone numbers were not available. One hundred and three workers (20%) were excluded because they no longer lived in the geographic area of interest (New Jersey, the Philadelphia metropolitan area, and southeastern New York). A total of 130 workers (25%) were ineligible (primarily because no child under age 6 was living in the home), and 47 eligible workers (9%) refused, leaving a total of 26 eligible workers from the ABLES registry who agreed to participate in the study. Three construction workers from a local union also participated in the study, for a total of 29 exposed households. Eighteen families nominated by the workers agreed to participate as neighborhood control households. A total of 59 children (37 exposed and 22 control subjects) were under age 6. Nine of these children (6 exposed and 3 control subjects) were subsequently excluded from the blood lead analysis because venous blood samples could not be obtained.

Workers had been employed in construction, on average, for 11.6 years (range 1 through 25 years). Half of the workers (50%) had received training about the danger of lead prior to starting their most recent construction job. Some of the workers were provided company-laundered work clothes (28.6%) and shower facilities (32.1%). Half of the workers (50%) reported changing out of work clothes prior to leaving work. Most workers (78.6%) wore at least some street

clothes at work, and almost all (90.9%) laundered these clothes at home. Almost half (46.4%) reported taking or wearing their work shoes home; only 17.9% reported always showering before leaving work. The majority of workers (75%) drove their personal vehicle to and from the work site.

Exposed homes were somewhat more likely than control homes to have household members who participated in a lead-related hobby (20.7% vs 5.6%, *P* = .23). Other factors, such as age of the home, remodeling during the past year, consumption of canned foods, and presence of a pet that spends time both indoors and outdoors, showed no differences between exposed and control homes. There were no differences between exposed and control homes in the percentage of lead in household paint or in the level of lead in drinking water.

Results of dust lead sampling in automobiles and homes are described elsewhere.⁸ Dust lead levels were significantly higher in the automobiles of construction workers than in those of control households and were generally higher in the homes of construction workers than in control homes.

Table 1 provides unadjusted blood lead results by exposure status for children under age 6. There was no evidence of confounding or effect modification by nonoccupational factors. Children in exposed homes (n = 31) had somewhat

higher mean blood lead levels than children from control homes ($n = 19$) (arithmetic mean 7.4 vs 5.1 $\mu\text{g}/\text{dL}$; $P = .11$). A greater percentage of exposed children than control children had blood lead levels that were at or over the CDC intervention level of 10 $\mu\text{g}/\text{dL}$ (25.8% vs 5.3%; $P = .13$). Exposed children were six times more likely than control children to have a blood lead level of 10 $\mu\text{g}/\text{dL}$ or greater (odds ratio [OR] = 6.1, 95% confidence interval [CI] = 0.9, 147.2). The confidence intervals are quite wide owing to the small study size. Adjustment for multiple members of the same household with the correlated-outcome method of Zeger and Liang¹¹ gave similar results in both linear and logistic models. There was no statistically significant difference between exposed and control children in mean level of erythrocyte protoporphyrin.

Workers were categorized by job title into those we considered to have high potential for home contamination (e.g., sandblaster, painter) and those we considered to have low potential for home contamination (e.g., ironworker, foreman), on the basis of the frequency and magnitude of exposure to lead dusts. We observed an increased risk for elevated blood lead in children of workers with high potential for home contamination ($n = 16$) compared with control children ($n = 19$) (OR = 7.1; 95% CI = 0.9, 187.4). Children of workers with low potential for home contamination ($n = 14$) had an odds ratio of 4.7 (95% CI = 0.4, 135.8). The confidence intervals for these estimates are wide and overlap. Children of workers who reported never, or only sometimes, changing out of work clothing before going home were somewhat more likely to have an elevated blood lead level (OR = 1.6; 95% CI = 0.3, 10.2) than were children of workers who reported always or usually changing out of work clothing.

Among exposed families, the child's blood lead levels were significantly correlated with dust lead levels at most sampling locations in the home and automobile, but correlations between child's blood lead and environmental measures were not found for control families. Of note for exposed homes were particularly strong correlations with lead dust levels at the main entry and in the family room (correlation coefficients = .49 and .58, respectively).

Discussion

Exposure to toxic materials originating in the workplace has been known in

the occupational health community to be a concern for families of workers in several industries. Outbreaks of severe illness caused by asbestos,¹² beryllium,¹³ and polycyclic compounds¹⁴ have been traced to home contamination by industrial dust. Lead is of particular concern for workers with young children since elevated blood lead levels have been implicated as a cause of a variety of health problems in children, ranging from behavioral disorders to brain damage.¹⁵⁻¹⁷ Children are at higher risk for lead exposure because they have more hand-to-mouth activity than adults, and the efficiency of gastrointestinal absorption of lead in children exceeds that in adults.¹⁸ There are numerous reports documenting home contamination with lead in general industry (e.g., lead smelter, battery recycling),¹⁹⁻²⁶ although many of these studies are limited by the method of blood collection (e.g., fingerstick) or by lack of a comparison population. This study is the first comprehensive examination of home lead contamination among construction workers.

We found that the children of lead-exposed construction workers were six times more likely to have a blood lead level of 10 $\mu\text{g}/\text{dL}$ or greater than the children of neighbors employed in non-lead industries. The higher levels of lead dust in the exposed homes compared with the controls⁸ and, among exposed children, the correlation of blood lead levels with environmental dust levels are consistent with dust lead contamination being the major exposure pathway. The importance of interior dust lead levels as a lead-exposure pathway has been identified for other children at high risk for lead poisoning, such as those living in older, urban housing.^{27,28}

The study is limited in size and representativeness. Because the majority of workers were identified through a lead registry, the children in the study may not be representative of the children of lead-exposed construction workers in general. However, the range of job titles and other employment characteristics of the study population suggest that these workers are not meaningfully different from other lead-exposed construction workers with elevated blood lead levels. The high percentage of workers that we were unable to contact during recruitment reflects the difficulty of studying this highly mobile population. Workers who were eligible for the study but who chose not to participate were similar to participants with respect to age and mean blood lead level.

The OSHA lead standard for construction was implemented in August of 1993,⁵ one year prior to study data collection. Prior to enactment of the standard, showers and change facilities were not provided at most construction sites.⁴ It is difficult to use our data to evaluate the effect of the new standard since lead contamination in workers' homes and automobiles may reflect accumulation over many months or years. Furthermore, many of the provisions of the lead standard that are intended to prevent lead dust from leaving the work site, such as change facilities and showers, are required only if a worker's airborne lead exposure exceeds the permissible exposure limit of 50 $\mu\text{g}/\text{m}^3$. Air-sampling measures, because they do not take into account other sources of exposure such as surface dust contamination, may not adequately predict levels of lead dust taken home from the workplace.²⁹ A survey of general industry practices revealed that even the required environmental and biological monitoring for lead is not routinely conducted.³⁰

For the most part, workers in the lead registry are individuals who (1) received routine blood lead monitoring at the workplace or (2) saw a medical provider who performed a blood lead test. Therefore, our findings suggest that some employers may be conducting routine monitoring but not putting measures in place to reduce home contamination, and/or that medical providers who screen occupationally exposed adults are not recommending screening of young children who live in workers' homes. In this light, our findings emphasize the importance of CDC's guidelines for preventing lead exposure in children. The CDC is revising its 1991 guidelines to recommend that children should be screened for lead if they live in a high-risk geographic area, belong to a high-risk population group (such as low-income children), or have individual risk factors for lead exposure (P. Briss, telephone conversation, 1996). Children of occupationally exposed workers are one high-risk population group for whom screening should be considered. Furthermore, although lead poisoning can be ameliorated when identified, the focus should be on primary prevention of exposure. Practicing physicians and local health departments should assess parental occupational exposures as a possible pathway for lead exposure to young children and should counsel parents on ways to avoid bringing lead dust home from work (e.g., through the use of

change and shower facilities at the work site). □

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References

- Centers for Disease Control. *Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control*. Atlanta, Ga: Public Health Service; 1991.
- Pirkle JL, Brody DJ, Gunter EW, et al. The decline in blood lead levels in the United States: the National Health and Nutrition Examination Surveys (NHANES). *JAMA*. 1994;272:284–291.
- Brody DJ, Pirkle JL, Kramer RA, et al. Blood lead levels in the US population: phase 1 of the third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991). *JAMA*. 1994;272:277–283.
- NIOSH Alert: *Request for Assistance in Preventing Lead Poisoning in Construction Workers*. Cincinnati, Ohio: Centers for Disease Control; 1991.
- Lead Exposure in Construction: Interim Final Rule. 29 CFR §1926 (1993).
- Standard Industrial Classification*. Washington, DC: Office of Management and Budget; 1972.
- Tepper A. Surveillance of occupational lead exposure in New Jersey: 1986 to 1989. *Am J Public Health*. 1992;82:275–277.
- Piacitelli GM, Whelan EA, Sieber WK, Gervel B. Elevated lead contamination in homes of construction workers. *Am Ind Hyg Assoc J*. 1997;58:447–454.
- LogXact-Turbo User Manual*. Cambridge, Mass: CYTEL Software Corp; 1993.
- StatXact-Turbo User Manual*. Cambridge, Mass: CYTEL Software Corp; 1992.
- Zeger SL, Liang K-Y. Longitudinal data analysis for discrete and continuous outcomes. *Biometrics*. 1986;42:121–130.
- Anderson HA, Lilis R, Daum SM, Selikoff IJ. Asbestosis among household contacts of asbestos factory workers. *Ann N Y Acad Sci*. 1979;330:387–399.
- Eisenbud M, Wanta RC, Dustan C, et al. Non-occupational berylliosis. *J Ind Hyg Toxicol*. 1949;31:282–294.
- Jensen NE, Sneddon IB, Walter AE. Chloracne: three cases. *Proc R Soc Med*. 1972;65:687–688.
- Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood. *N Engl J Med*. 1990;322:83–88.
- Mushak P, Davis JM, Crocetti AF, Grant LD. Prenatal and postnatal effects of low-level lead exposure: integrated summary of a report to the U.S. Congress on childhood lead poisoning. *Environ Res*. 1989;50:11–36.
- Toxicological Profile for Lead*. Atlanta, Ga: US Public Health Service; 1990.
- Ziegler EE, Edwards BB, Jensen RL, et al. Absorption and retention of lead by infants. *Pediatr Res*. 1978;12:29–34.
- Baker EL, Folland DS, Taylor TA, et al. Lead poisoning in children of lead workers: home contamination with industrial dust. *N Engl J Med*. 1977;295:260.
- Rice C, Fischbein A, Lilis R, et al. Lead contamination in the homes of employees of secondary lead smelters. *Environ Res*. 1978;15:375–380.
- Winegar DA, Levy BS, Andress JS Jr, et al. Chronic occupational exposure to lead: an evaluation of the health of smelter workers. *J Occup Med*. 1977;19:603–606.
- Morton DE, Saah AJ, Silberg SL, et al. Lead absorption in children of employees in a lead-related industry. *Am J Epidemiol*. 1982;115:549–555.
- Watson WN, Witherell LE, Giguere GC. Increased lead absorption in children of workers in a lead storage battery plant. *J Occup Med*. 1978;20:759–761.
- Dolcourt JL, Hamrick HJ, O'Tuama LA, et al. Increased lead burden in children of battery workers: asymptomatic exposure resulting from contaminated work clothing. *Pediatrics*. 1978;62:563–566.
- Matte TD, Figuerroa JP, Ostrowski S, et al. Lead poisoning among household members exposed to lead-acid battery repair shops in Kingston, Jamaica. *Int J Epidemiol*. 1989;18:874–881.
- Gittleman JL, Engelgau MM, Shaw J, Wille KK, Seligman PJ. Lead poisoning among battery reclamation workers in Alabama. *J Occup Med*. 1994;36:526–532.
- Bornschein RL, Succop P, Dietrich KN, et al. The influence of social and environmental factors on dust lead, hand lead, and blood lead levels in young children. *Environ Res*. 1985;38:108–118.
- Clark S, Bornschein R, Succop P, et al. Urban lead exposures of children in Cincinnati, Ohio. *Chem Speciation Bioavailability*. 1991;3:163–171.
- Piacitelli GM, Whelan EA, Ewers LM, Sieber WK. Lead contamination in automobiles of lead-exposed bridgeworkers. *Appl Occup Environ Hyg*. 1995;10:849–855.
- Rudolph L, Sharp DS, Samuels S, Perkins C, Rosenberg J. Environmental and biological monitoring for lead exposure in California workplaces. *Am J Public Health*. 1990;80:921–925.